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Case report

Speed of development of cerebral swelling following blunt cranial trauma

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ABSTRACT

A 22-year-old male suffered severe injuries to the head, chest and abdominal cavities in a vehicle crash, with death occurring at the scene. At autopsy, the cranial cavity was opened and markedly disrupted with compound and comminuted fracturing of all bones of the skull and facial skeleton. The brain showed extensive lacerations with almost complete parenchymal disruption. However, a preserved fragment of right frontal lobe exhibited marked swelling with gyral flattening. This case could provide further evidence for prompt cerebral swelling after blunt head trauma, and is supportive of animal studies that have demonstrated rapid swelling that is most likely is related to reactive vasodilation rather than to vasogenic oedema.

1. Introduction

The speed with which swelling of the brain occurs following blunt craniocerebral trauma has been debated amongst forensic pathologists, with suggestions that it may be a rapid process contrasting with other opinions that it may on occasion occur much more slowly.[1] A case is reported where lethal head and other injuries resulted in rapid death but where focal gyral flattening of the brain was observed suggesting an almost immediate cerebral response.

2. Case report

A 22-year-old male on a motor-cycle collided at high speed with a street sign and was thrown onto the road in the path of an oncoming vehicle. He was declared dead at the scene. At autopsy there were major injuries to the chest and abdominal cavities with lacerations of the heart and aortic transection. There were also significant injuries to the head with disruption of the cranial cavity and compound and comminuted fracturing of all bones of the skull and facial skeleton (Fig. 1). The brain showed extensive lacerations and parenchymal disruption. Of note, a preserved fragment of right frontal lobe demonstrated marked swelling with gyral flattening (Fig. 2). A close up picture of the area of gyral flattening is shown in Fig. 3A compared to a normal control in Fig. 3B.

There were no underlying organic diseases present which could have caused or contributed to death and the injuries were compatible with the described incident. Toxicology screening was negative. Death had been caused by multiple blunt force injuries.

3. Discussion

Previous investigations into changes in intracranial pressure (ICP) in an animal model have shown that a rise in ICP occurs within minutes after inflicted blunt craniocerebral trauma in a laboratory situation, and correlates with systemic hypertension.[1-3] The response to blunt trauma appears to be biphasic with an initial rapid response that shows gradual diminution, followed by a steady increase. We consider that the early response is most likely due to reactive vasodilatation given that oedema takes some time to develop after an injury.[4,5] Immunohistochemical staining of brain sections in impacted animals has confirmed oedema by showing intense staining for albumin 4 h after trauma due to breakdown of the blood–brain barrier, with leakage of albumin–rich serum into the interstitium.[1] Unfortunately we have not been able to successfully replicate this finding in human post-mortem material due to

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Fig. 1. Completely disrupted cranial cavity after opening at autopsy demonstrating comminuted compound fracturing of all major skull bones involving all of the cranial fossae.

nonspecific background staining associated with post-mortem tissue degradation. An important point in the animal model is that at no time did the ICP or associated cerebral oxygenation approach normal values. We theorize that significant blunt head trauma causes an immediate increase in ICP due to reactive vasodilation in the short term, which is then reduced slightly by compensatory mechanisms, and is then compounded by vasogenic oedema.[3] The significance of this in forensic terms is that individuals who suffer severe blunt cranial trauma which leads to death (not involving the slowly developing extradural haematoma) are unlikely to exhibit normal behaviour after impact, given that intracranial pressure increases rapidly with the associated reduction in arterial perfusion and brain parenchymal oxygenation.

In humans the speed of brain swelling has been shown to be quite rapid on cerebral imaging with computerized tomography (CT) showing cerebral swelling as early as 1 h and 17 min following trauma.[6] Clinical studies are, however limited by the time in which it takes to bring an injured individual to a hospital, to be stabilized and then to be available for radiological studies. The advantage of forensic cases is that the findings at the time of death may be 'frozen in time'. In the reported case, the decedent was observed to be travelling on a motor cycle at high speed prior to colliding with a fixed object, hitting the road and then being impacted by another vehicle. At autopsy there was evidence of lethal deccelerative injury in the form of a transected aorta, in addition to other multiple and serious injuries to the chest, abdominal cavity and head. Of note, despite the extensive and obviously immediately lethal injuries (Fig. 1) with marked traumatic disruption of the brain, the remaining intact portions of cerebral cortex showed obvious gyral flattening in keeping with brain swelling (Figs. 2 and 3). Given that it is unlikely that there would have been time to develop significant interstitial oedema in the seconds between impact, injury and death, the most likely cause of the gyral flattening was reactive vasodilation. Vasodilation and hypereamia have certainly been demonstrated within 30 min of trauma.[7,8] However, even if the exact mechanism is uncertain it appears that brain swelling may develop extremely rapidly following blunt trauma, in keeping with the findings of animal experiments.

The possibility of a direct compressive effect from other parts of the brain was considered in the reported case, however shattering of the skull meant that the cranial cavity was no longer a closed compartment and so was essentially already decompressed. In addition, similar gyral flattening has been observed by the authors

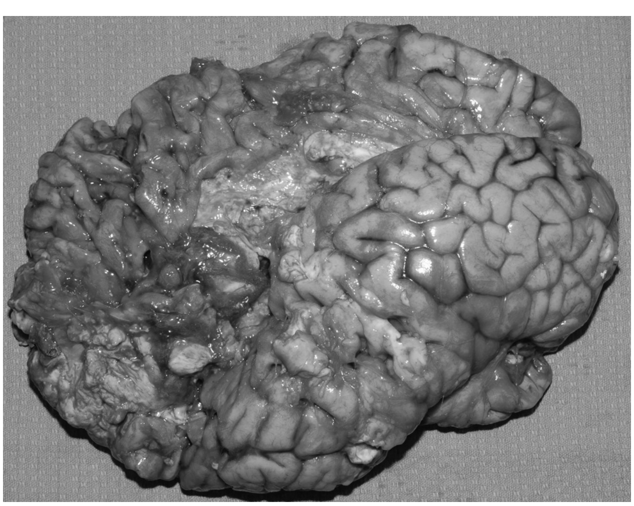

Fig. 2. Massively traumatised brain showing laceration and parenchymal disruption with preservation of a portion of the right frontal lobe. In this area there is obvious gyral flattening from cerebral swelling. Although it is more severe towards the centre of the lobe, all gyri showed some degree of flattening, as can be better appreciated in Fig. 3A.

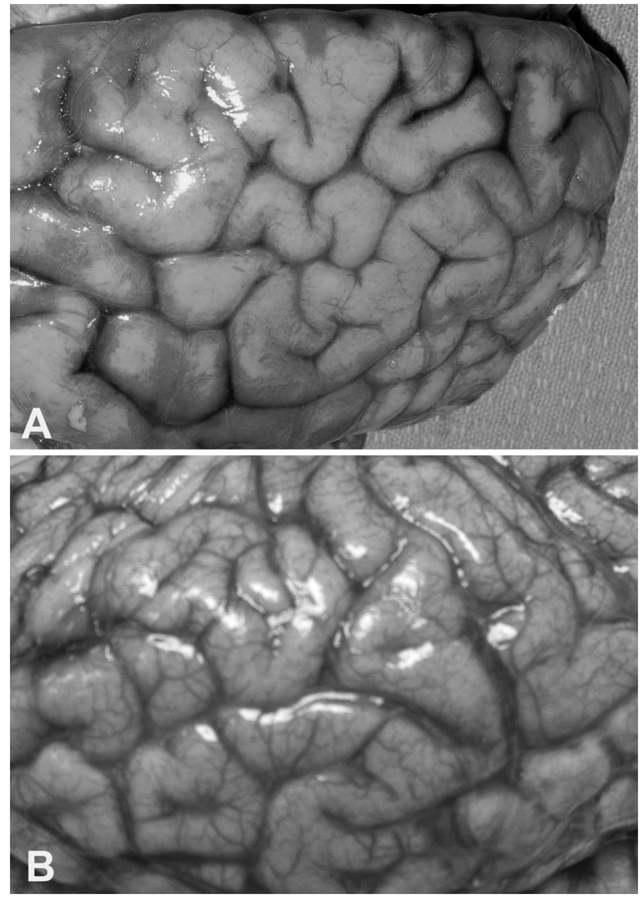

Fig. 3. A higher power view of the area of cerebral swelling (A) compared to a normal control without swelling (B).

in other motor vehicle crash victims with lethal atlanto-occipital fracture dislocations and no significant space occupying intracranial haemorrhage or parenchymal disruption. Both of these points would be in keeping with a local vasoreactive basis for the observed gyral flattening. The other possibility of chest compression with a rapid rise in intravascular pressure was also considered less likely given that lacerations of the aorta and heart would quickly depressurise the extracranial vasculature.

The current case therefore provides an example of cerebral swelling immediately following blunt head trauma in a human model. While it is possible that cerebral swelling may develop more slowly in some cases, this case and previous animal studies suggest that ICP changes may be extremely rapid. In the case of rapid increases in ICP, an immediate alteration in conscious state would be likely due to concomitant reduction in cerebral perfusion pressure and brain oxygenation, without a period of clinical normality.

Ethical approval

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None.

Conflict of interest

None.

References

1. Byard RW, Bhatia K, Reilly P, Vink R. How rapidly does cerebral swelling follow trauma? Observations using an animal model and possible implications in infancy. *Leg Med (Tokyo)* 2009 April;**11**(suppl. 1):S128–31.
2. Vink R, Bhatia KD, Reilly PL. The relationship between intracranial pressure and brain oxygenation following traumatic brain injury in sheep. *Acta Neurochir Suppl* 2008;**102**:189–92.
3. Byard RW, Gabriellian L, Helps SC, Thornton E, Vink R. Further investigations into the speed of cerebral swelling following blunt cranial trauma. *J Forensic Sci* 2012;**57**:973–5.
4. Barzo P, Marmarou A, Fatouros P, Hayasaki K, Corwin F. Contribution of vasogenic and cellular edema to traumatic brain swelling measured by diffusion-weighted imaging. *J Neurosurg* 1997;**87**:900–7.
5. O'Connor CA, Cernak I, Vink R. The temporal profile of edema formation differs between male and female rats following diffuse traumatic brain injury. *Acta Neurochir Suppl* 2006;**96**:121–4.
6. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Neglect* 1997;**21**:929–40.
7. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema". *J Neurosurg* 1981;**54**:170–8.
8. Yoshino E, Yamaki T, Higuchi T, Horikawa Y, Hirakawa K. Acute brain edema in fatal head injury: analysis by dynamic CT scanning. *J Neurosurg* 1985;**63**:830–9.